

Clinical Therapeutics

allele A in the study group was 8%, in the control group – 4% ($p = 0.1$; chi-square). The following genotypes distribution was observed in the study group: AA – 0.02; AG – 0.13; GG – 0.85. In the control group the following genotype distribution was observed: AA – 0.01; AG – 0.05; GG – 0.94. Distributions corresponded to Hardy-Weinberg equilibrium. **Conclusion:** In our prospective study, we observed tendency to genetic predisposition to higher activity of CYP3A5 in women with stillbirth compared with matched women with normal pregnancy. The results, however, did not reach statistical significance, which may demonstrate either lack of real association or insufficient number of subjects recruited. The observation needs to be proved or disproved in a larger population. **Disclosure of Interest:** None declared.

PP153—EVALUATION OF THE RELATIONSHIPS BETWEEN ABCB1 C3435T AND G2677T/A POLYMORPHISMS AND CLINICAL RESPONSE TO VENLAFAXINE IN PATIENTS WITH MAJOR DEPRESSIVE DISORDER

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Introduction: Venlafaxine, as a substrate of p-glycoprotein, is a widely used serotonin-noradrenaline reuptake inhibitor (SNRI). The aim of the study is to investigate the influence of ABCB1 G2677T/A, C3435T polymorphisms on efficacy of venlafaxine.

Patients (or Materials) and Methods: Patients ($n = 52$) who met the *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition-IV* criteria for major depressive disorder (MDD) were enrolled the study. All patients had affirmed for once a day administration of venlafaxine at 8:00 to 9:00 AM during the study. Protocol visits were completed at baseline, 1st, 2nd, 4th, and 6th weeks. The clinical response to venlafaxine was evaluated by psychiatrists with 17-item Hamilton Rating Scale for Depression (HAM-D17). Blood samples were taken for genotyping at 4th week of the study. Genotyping for the ABCB1 gene 3435C>T and G2677T/A polymorphisms was performed by PCR/RFLP assays.

Results: Our results showed that there is no correlation between efficacy and tolerability of venlafaxine and ABCB1 G2677T/A, C3435T polymorphisms. But carriers of the TT genotype for 3435C>T polymorphism and carriers of the TT/TA genotype for G2677T/A polymorphism could be tended to be poor responder (Table).

Table. HAM-D₁₇ scores according to ABCB1 G2677T/A, C3435T genotypes.

	3435C>T		2677G>T/A	
	CC-CT 41	TT 11	GG-GT-GA 41	TT-TA 11
n				
HAMD ₁₇ baseline	21.56 ± 0.695	22.45 ± 2.077	21.78 ± 0.737	21.64 ± 1.865
HAMD ₁₇ 1st week	14.93 ± 0.851	16.73 ± 2.195	15.34 ± 0.852	15.18 ± 2.252
HAMD ₁₇ 2nd week	13.76 ± 0.939	15.64 ± 1.820	13.59 ± 0.907	16.27 ± 1.978
HAMD ₁₇ 4th week	11.27 ± 0.968	11.54 ± 1.883	11.24 ± 0.931	12.36 ± 2.125
HAMD ₁₇ 6th week	9.63 ± 1.11	10.09 ± 2.095	9.54 ± 1.038	10.45 ± 2.577
	F = 0.41, P = 0.741		F = 0.672, P = 0.563	

Conclusion: Although our results showed that there is no correlation between efficacy of venlafaxine and ABCB1 G2677T/A, C3435T polymorphisms, we couldn't reach the sufficient patient number. There is need for studies with sufficient patient number and haplotype analysis including also ABCB1 C1236T polymorphism in MDD patients.

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PP154—PERSONALISING HEALTH CARE: FEASIBILITY AND FUTURE IMPLICATIONS FOR ALL STAKEHOLDER GROUPS INCLUDING AUTHORITIES, PHYSICIANS AND PATIENTS

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Introduction: The promise of personalized care has not always translated into improvements in patient care. There are concerns among payers that advice for certain genetic tests has been revoked, diagnostic tests can be costly, and there is fragmentation of funding of care including tests. In addition, pharmaceutical companies are seeking high prices for new targeted drugs through designating them as orphan drugs. Consequently, there is a need to integrate current knowledge about the value of genetic, biomarkers, prognostic tests and targeted drug therapies from a health authority perspective to provide future guidance.

Patients (or Materials) and Methods: This will be achieved by (1) reviewing the current literature regarding personalized medicine; (2) appraising key funding, organizational, and health care issues that need to be addressed especially from a health authority perspective; and (3) suggesting future avenues for all key stakeholder groups to enhance future funding and utilization of new personalized approaches to improve future patient care. The latter will be achieved through an iterative process.

Results: Multiple findings are consolidated under headings. These include (1) general considerations incorporating definitions and the need for different approaches to progress personalized medicine; (2) knowledge about the influence of pharmacogenomics on response and toxicity of drug therapies using current examples including cases where recommendations have recently been revoked; (3) knowledge of the value of biomarker tests to target treatment approaches; (4) challenges and concerns including the potentially high cost of tests and targeted therapies and current fragmentation of funding; and (5) key issues for health care funding bodies to address to enhance funding for new diagnostic/prognostic tests as well as new targeted therapies. Guidance is given on potential ways forward for all key stakeholder groups including reviewing key medical, ethical, legal,